CHANGES IN ELECTROENCEPHALOGRAM SPECTRA DURING REPEATED EXPOSURE TO $^{+}$Gz ACCELERATION

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13. ABSTRACT

Eight human subjects experienced a series of +Gz acceleration profiles reaching peak values of +4½ Gz and +6 Gz. Each subject experienced six 45-second +Gz exposures within 15 minutes. Electroencephalograms (EEG) were made throughout this period and subjected to a narrow-band spectral analysis within 10-second epochs. The subjects wore protective G-suits, and did not experience impairment of central vision during the runs analyzed. Small increases in spectral intensity of the EEG were seen during eyes-closed, resting epochs following +Gz exposure. These increases did not change overall shape of spectral profiles and did not exceed normal levels of EEG intensity. During +Gz forces with eyes open, the raw EEG was dominated by electromyographic (EMG) activity, which was typically proportional to instantaneous +Gz force but might also reflect paroxysmal whole-body contractions occurring at scattered points in the +Gz exposure profile. Increased EEG intensities greatly exceeding basal levels for certain narrow frequency bands were observed during actual +Gz exposures. In all subjects, a return to normal EEG spectral intensity profiles took place within 30 seconds of termination of acceleration. Although +6 Gz exposure was a notably more severe stress than +4½ Gz, no cumulative effects were noted across six exposures in any sequence. Similar rapid returns to base levels were noted in an additional subject exposed to +7 Gz for continuous periods of 45 seconds.

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FOREWORD

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This technical report has been reviewed and is approved.

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SECTION I

INTRODUCTION

Visual blackout and lapse of consciousness can occur during transient exposures to acceleration in the +4 to +6 Gz range. These responses are primarily due to retinal anoxia following collapse of the retinal artery, and to cortical anoxia following impairment of circulation above the carotid artery. Impairment of consciousness is regularly accompanied by electroencephalographic (EEG) changes indicative of pathology, and both the depth and duration of lapses of consciousness can be determined with precision from the EEG record (ref. 22).

With the use of anti-G protective devices (inflating G-suits) consciousness can be maintained during transient exposures exceeding +6 Gz. A number of studies reviewed by Grether (ref. 13) and Fraser (ref. 10) have raised the suggestion that central nervous processes are impaired by +Gz acceleration in the 4–6 G range, even when G-suit protection against complete visual blackout is provided. Repeated subacute anoxia can have cumulative effects within a moderately long (10–20 min) epoch, and +Gz-contingent mechanical deformation of brain tissue can lead to pressure on gross hippocampal and midbrain structures and mechanical deformation of cell ultrastructure and synapses (refs. 15, 19). All these effects should be reflected in the spectral characteristics of the concurrent EEG.

This report describes an electroencephalographic evaluation of nine subjects exposed to repeated +Gz acceleration in the 4.5–6 G range without loss of consciousness. We have attempted to evaluate EEG characteristics during the accelerations themselves, during resting epochs immediately post and prior, and during long-term (20 min) epochs incorporating different sequences of +Gz exposure levels. We attempted to determine whether any indications of central nervous system (CNS) impairment were present even though loss of consciousness did not occur.

A number of prior studies have described the EEG characteristics of blackout and loss of consciousness during +Gz acceleration, both in centrifuges and actual flight (refs. 5, 18, 21, 22). The classic EEG signs of anoxia (delta waves) and of general transient pathology (theta waves; paroxysmal foci) have been reported in records made both during and after acceleration. These signs are gross and can be seen in unprocessed paper tracings of the EEG even at levels below full unconsciousness (ref. 17). When more subtle EEG changes are sought, which might be diagnostic of central nervous impairment without blackout or attenuation of consciousness, three difficulties arise:

1. The relevant EEG features may not be in the form of sinusoidal waves or spindles, but in change of intensity in the different EEG frequency bands. Visual inspection is inadequate and some form of spectral analysis must be employed.

2. A normal baseline of spectral intensities is difficult to define experimentally, since both transient changes in levels of arousal and central nervous responses to stress cause EEG changes of the same order of magnitude and in the same frequency bands as does exposure to +Gz forces.

3. The presence of signals of electromyographic origin combined with electrode contact artifacts due to head movements inevitably obscures the EEG traces during movements of peak acceleration (figure 1).[1]

In this study we have attempted to minimize these difficulties by using motion-artifact resistant electrodes, employing a balanced design of +Gz exposures with many resting epochs for reference levels, and using a digital, narrow-band form of spectral analysis which minimizes the infiltration of myoelectric signals into certain portions of the EEG spectrum. Depth electrode studies on animals (refs. 2, 3) in which EMG infiltration was theoretically minimal, serve to compare and confirm the EEG characteristics derived from spectrally analyzed scalp activity.

A study of narrow-band myogram spectra derived from heavily contracted scalp musculature in humans provided a baseline for anticipating EMG intensity levels within the EEG frequency band during acceleration (ref. 20). By comparing ratios of intensities between different frequency bands during the evolution of a +Gz exposure profile, a logical methodology of segregating neuroelectric from myoelectric intensity shifts was developed. When evaluated in this fashion the EEG serves as a sensitive indicator of subclinical pathology, transient CNS impairment and stress (ref. 1).

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[1] Figures and tables are located at end of report.

SECTION II

PROCEDURE

The centrifuge located at Brooks Air Force Base, Texas was used to produce the acceleration profiles described herein (see ref. 16). All subjects had previous centrifuge experience, and all were medically qualified to undergo exposure to increased G forces. Subjects' ages were between 23 and 37 (mean, 26.7). Nine males and one female were used in the studies reported here.

For EEG recording, Beckman biopotential miniature electrodes injected with Offner paste were attached to the shaved, burred skin at the P3 to 01 and P4 to 02 sites (ref. 14), and a ground electrode was placed on the mastoid bone. Resistance between homolateral electrodes ranged from 0.6 to 2.5 K ohms (d-c resistance) immediately after application. These electrodes provide a buffered skin-jell-electrode-silver chloride-silver interface that resists polarization, and hence minimizes movement artifacts. For EKG recording, Beckman biopotential electrodes were attached to sternal and biaxilliary positions. During all runs, subjects wore the CSV 12/P anti-G garment which was activated at +2 G and produced 2 lb/in^2 at 2 G and 1½ lb per G above 2 G.

After the subject entered the gondola and all calibrations had been obtained, a series of general instructions was read over the intercom system. Subjects were not permitted to know the number or sequence of runs, although they had been informed that the experiment would involve several exposures to a maximum of +6 Gz. They were told to relax as much as possible during the exposure, but to strain if they felt themselves approaching blackout.

The sequence of events for a given exposure involved several distinct periods designed to produce differing states of physiological activation. At the start of the sequence, the subject was instructed to sit back with eyes closed, and relax. This produced 15 seconds of "resting" EEG records. At the end of this period, the centrifuge operator went through his checklist and obtained a final ready signal from the subject. He then announced that the brake was off. The operator then waited 15 to 20 seconds before initiating acceleration. During this period, the subject anticipated the start of the run with eyes open and, thus, a period of "activated' EEG was obtained. A total of 15 seconds was taken to accelerate linearly up to the desired G level for each run. This peak level was maintained for 15 seconds, and then 15 seconds were spent in deceleration to reach a total stopping of the centrifuge. Thus, each run was 45 seconds long and consisted of three identifiable periods—increasing G, peak G, and decreasing G.

After the centrifuge was stopped, a silence was maintained for 15 to 20 seconds while the subject remained with eyes open. This produced a postrun period of activated EEG. Finally, after reporting the presence or absence of visual symptoms, the subject was instructed to relax with eyes closed again for 15 seconds of "resting" records. These events constituted one "run" for a subject. Each run defined seven epochs, four resting and three active, which are schematically outlined in figure 2.

For each subject, six "runs" were obtained sequentially. For three consecutive runs, the peak level reached was +4.5 Gz; for the other three runs, the peak was +6 Gz. In five subjects, the 4.5 G runs came first; in the other four subjects, the 6 G runs came first. The sequence of runs, either 4.5→6 or 6→4.5, were assigned alternately as each subject reported for the experiment according to availability. During the +Gz exposure, subjects were instructed to monitor two green peripheral signal lights, one central red light, and to come off a dead-man's switch if any loss of central vision was noted. This automatically terminated the run. Runs were also terminated if significant cardiac pathology was noted by the medical monitor. Aborted runs were included in the EEG analysis protocol if a minimum of 5 seconds at peak G load was available. If 5 seconds was not available or if one or more runs were cancelled by the medical monitor, the subject was excluded from the experiment.

The +Gz runs were a distinct stress to our subjects, particularly when occurring after three +4½ Gz exposures. Three of five subjects exposed in this sequence aborted one or more +6 Gz runs due to central vision impairment. One subject was disqualified by the medical monitor for cardiac abnomalities after one +6 Gz run, and another subject (No. 5) showed some cardiac arrhythmia, but not sufficient to disqualify. By contrast, no subjects exposed to +6 Gz in the first three runs aborted a run, or exhibited cardiac abnormalities. We considered the subjects exposed to +6 Gz during runs 4–6 to have been under severe cardiac stress during the peak G force and to have all suffered complete impairment of peripheral vision. Those exposed to +6 Gz during runs 1–3 were apparently under a slightly less severe stress and did not report total loss of peripheral vision. No cardiac symptoms were reported during any +4½ Gz runs, and the visual impairment reported was limited to slight dimming of the peripheral lights, usually compensated by straining.

A video monitor system enabled the investigators to observe a full face view of the subjects throughout each run. Head and eye movements and general body distortions and straining were noted, and collated with artifacts appearing in the EEG tracings. For two subjects a videotape recording of the six-run sequence was made, and head and eye movements could be evaluated during slow-motion playback.

Data analysis of the EEG records was accomplished on the IBM 360/91 computer located at the Health Sciences Computing Facility, UCLA. For each epoch of interest, a relatively artifact-free period of 10 to 15 seconds duration was digitized at 256 samples/sec using the DPL facility of the Brain Research Institute, UCLA. Power spectra were then calculated for each epoch using the BMD X-92 program (ref. 8), and printed out as cumulated power in five separate bands. In this way, a balanced group of eight subjects was obtained in which the effects of the two sequences (4½→6 vs 6→4½) could be determined. Of this group, two subjects were exposed to higher G-forces (+6½ and +7 Gz, respectively) for over 45 seconds and EEG material was recorded over the subsequent 20-minute period, to detect any slowly evolving EEG changes contingent on trauma to CNS tissues.

SECTION III

INTERPRETATION OF EEG POWER SPECTRUM ANALYSIS

In the outline of EEG spectral band characteristics below, only power spectral density is considered, and the banding nomenclature does not refer to the sinusoidal "waves" of the same names that are often apparent during visual inspection of paper EEG tracings. The levels of intensity given are characteristic of the bipolar parietal-occipital derivation used in this study.

EEG intensities are reported independently for each band, and not as proportions of total power. The units used are microvolts^2/per second/per hertz (ref. 23), so that numbers may be compared or averaged directly across epochs without regard to bandwidth or irregular epoch lengths. The banding used is based on the division of the EEG spectrum into alpha, beta, delta and theta regions, each representing the output of nominally independent physiological processes in the waking state (ref. 6) and each differentially sensitive to EMG infiltration (ref. 20).

DELTA ACTIVITY, 1–3 Hz

Our digital filter window is sharply attenuated below 1 Hz, and does not admit activity reflecting baseline drift or slowly spreading global activity such as contingent negative variation. The normal resting levels of delta activity is an occipital-parietal derivation range from 5 to 20 μv^2/sec/Hz. This frequency band incorporates many electrooccular (EOG) phenomena, such as lateral scanning, eye movements and electromechanical events such as scalp distention due to eye blink and head movements. Scalp EMG infiltration is minimal in this frequency domain, but whole-body vibration due to widespread thoracic muscle contraction is readily apparent. Pathological activity due to anoxia and edematous tissue compression is also partially represented in this domain. EOG, mechanical and pathological events are usually of high intensity relative to normal resting levels, often reaching values exceeding 100–200 μv^2/sec/Hz and cannot be distinguished from each other without visual inspection of the record. However, a rapid return to resting intensity levels following exposure to stress is evidence for an absence of pathological processes even if the visible record is not interpretable due to superimposition of other events.

THETA ACTIVITY, 4–7 Hz

The resting levels of this activity are quite low, usually under 10 μv^2/sec/Hz. Bursts or spindles of theta activity have been associated with perceptual events (ref. 1). Continuously coherent homolateral theta is often indicative of transient stress. Subacute anoxia causes slowing of the dominant EEG rhythms into the 4–7 Hz band. Scalp EMG is not reflected in the theta band during moderate jaw and brow tension, but 5 to 10 μv^2/sec/Hz of EMG infiltration may be noted in parietal-occipital leads during brief episodes of extreme contraction. The bursting, spindling and slowing described above are all of higher intensity, usually in the 20–40 μv^2/sec/Hz range.

ALPHA ACTIVITY, 8–12 Hz

This domain includes the dominant sinusoidal "rhythms" often seen in resting paper tracings. These rhythms vary greatly in intensity during rest, both from subject to subject and from moment to moment. They are usually, but not necessarily attenuated when the eyes are opened, and their almost total abolition is a part of the EEG signature of "arousal" or "alerting." Strong, eyes-closed alpha activity is in the 50–100 μv^2/sec/Hz range. An "aroused" record may generate as little as 2 μv^2/sec/Hz of activity in this frequency domain. EMG infiltration may be considerable, but will rarely exceed resting levels of dominant alpha rhythms. Paradoxically, events that generate EMG activity invariably serve to attenuate a dominant alpha rhythm, so that signals of direct muscle origin may replace signals of cerebral origin with little change in the overall intensity envelope. However, EMG activity superimposed on the alert record is visually interpretable as such. No particular patterns of intensity in this band are interpreted as pathological in waking normal subjects.

BETA ACTIVITY, 13–20, 21–32 Hz

Bursting or spindling in the 13–20 Hz band is associated with both stress and perception (ref. 7). Pathological processes connected with both anoxia and reduced levels of consciousness of other origins also enhance intensity in this range. Resting activity is normally under 5 μv^2/sec/Hz in the 13–20 Hz band, under 2 μv^2/sec/Hz from 21–32 Hz. This activity is unchanged by opening and closing of the eyes.

EMG activity is well represented at these frequencies, and can exceed 50 μv^2/sec/Hz during jaw clenching alone. During spasmic whole body contraction, values ten times this high are possible. The enhanced muscle "tone" following contraction episodes is reflected in the 21–32 Hz band, as are occasional single-fiber spikes from scalp muscles. No quantified pathological or perceptual effects have been determined for the 21–32 Hz band in the right or left parietal-occipital derivations.

TEMPORAL-PARIETAL DERIVATIONS

The behavior of delta and theta activity is similar to parietal-occipital events. Eye movements are represented at higher intensities in the delta band. Theta activity of stressful origin may be more prominent, and high coherence between this activity and the parietal-occipital leads may be correlated with transient fluctuations in stressor effects.

Resting, eyes-closed temporal alpha is less prominent and occurs in fewer people than does occipital alpha. Sporadic alpha activity with eyes open is likely to be related to a relaxation of attention and incipient drowsiness. Alpha bursts are occasionally synchronized with the visual and auditory perception of of stimuli.

Intensity in the 13–20 Hz band is related to levels of attention, and homolateral coherence of beta activity fluctuates in response to changes in these levels. Shifts in beta coherence are also noted to accompany searching and scanning eye movements.

LATERAL DIFFERENTIATION OF EEG INTENSITIES

Delta activity rarely varies from side to side of the head unless pathological activity is present. Electrical activity of ocular and mechanical origin is almost always bilaterally represented.

Theta spindling, alpha activity of all kinds, and beta activity in the 13–20 Hz range is often laterally disproportionate. These effects are significant if of pathological origin, but reflect a differential functional organization of the brain when observed in response to stressor and perceptual events. No general principles for interpreting such differences can be given out of context.

SECTION IV

RESULTS
(EEG POWER INTENSITY LEVELS)

Numerical values of EEG spectral intensity, expressed in μv^2/sec/Hz, are listed in tables 1–13 for the various experimental epochs and frequency bandwidths defined in this study. Results are tabled separately for eyes-closed/resting and eyes-open/acceleration conditions. Eyes-closed/resting data is tabled separately for each frequency band; eyes-open/acceleration data is tabled separately for each subject. Unless otherwise noted, the tables give numerical averages of three runs for the left parietal-occipital channel. Since there was considerable variation in intensity levels within subjects, there was not sufficient data in most cases to establish statistical significance for differences between pairs of three-run means. The direction of change between epochs I and VII for each run is indicated in tables 1 to 5 by the symbols +, −, and =, and we use the term "consistent" to describe a shift in intensity of at least 10% occurring on each of three runs.

The balanced-exposure experimental protocol was designed to answer two questions: (1) are there any changes in the resting EEG during a sequence of exposures to +Gz forces, and (2) is the sequence of moderate (+4½ Gz) and high level (+6 Gz) exposures a factor in such changes as are observed? These questions may be answered by comparison of spectral characteristics of epoch VII vs epoch I (resting, eyes closed, 20 seconds before and after) and epoch VI vs epoch II (resting, eyes open, immediately before and after runs). This constituted the statistically controlled, "closed protocol" portion of our study.

In addition, EEG activity during the individual runs themselves was examined in an "open protocol" manner, and a descriptive evaluation of spectral characteristics was prepared. The two 45-second exposures to higher G levels were also treated descriptively, and the subjects were not provided with experimental controls.

EYES-CLOSED/RESTING DATA

Delta (1–3 Hz) Band (Table 1)

Visual examination of the EEG tracings failed to reveal any of the classical pathological signs based on slow waves, paroxysmal events or other visible patterns affecting 1–3 Hz intensity. With the exception of S3, all Ss displayed delta activity within normal limits. Elevated values of delta intensity following +Gz exposure were noted for S8 after all runs, and for Ss 3, 4, 6 and 10 after +6 G runs only. Ss 5, 9 and 11 showed no average pre-post run differences. Observed differences ranged from 2 to 20 μv^2 in intensity and from 33% to 80% of pre-run baselines. The elevation of postrun intensity after +6 G occurred consistently (10 of 12 runs) when the 6 G runs were experienced first, and only 7 of 12 runs when the 6 G runs were last, and with smaller percentage increases.

Theta (4–7 Hz) Band (Table 2)

Five of eight subjects showed a consistent increase in theta intensity postrun. Subjects 5, 11 and 10 did not. The levels of increase were small, and for all subjects but 3 and 4, theta values were in the "low background" category. Even in Ss 3 and 4, the theta intensities were derived from a complex signal, and sinusoidal "theta waves" were never seen. Few Ss exhibited a consistently higher level of theta activity bracketing +6 G exposures as compared to +4 G; and for the three who did, the differences were small (S 9, +1 μv^2, 25%; S 4, +2 to +5 μv^2, 20–50%; S 10, ½ μv^2, 20 %), and not related to the sequence of +Gz exposures.

Alpha Band (Table 3)

Alpha activity levels with eyes closed were indicative of the relative arousal or relaxation states of the subjects at various points in the experimental protocol, and intensities differed from individual to individual more than in other frequency bands. The changes observed between runs could not be readily classified into broad groups, and are related here for each subject individually. A comparison of spectral intensities with the visible character of alpha waves in the paper tracing is included.

Subject 3

Very high levels of alpha intensity, well-formed high amplitude alpha waves in the paper records. No differences after either +4½ G or +6 G runs. Relaxed, resting record at all times.

Subject 4

Moderate levels of alpha intensity, representing sporadic alpha "spindling" interspersed with prominent 15–20 Hz spindles peculiar to this subject. No differences between 4½ and 6 G runs were present in the postrun epochs, but some alpha blocking was noted prior to the 4½ G runs (runs 4–6).

Subject 5

The dominant frequency with eyes closed was a mixture of 12–14 Hz waves of low amplitude, which were attenuated upon arousal. Slight transient blocking of this activity was noted following runs 1–3 at 4½ G, and, more severely, preceding runs 4–6 at 6 G.

Subject 6

Very low level records at all times, no consistent changes noted between runs. Alert, aroused state at all times with alpha spindles noted at the moments of eye closure only. Background levels are slightly higher pre and post 6 G runs.

Subject 8

Strong alpha activity, with continuous sinusoidal alpha waves, postrun. Transient alpha block-

ing was noted in pre-run epochs, more often prior to runs 1–3 at 6 G. The observed increase in alpha intensity postrun is due to the suppression of this alpha blocking.

Subject 9

Moderate amplitude alpha intensity, some relaxed spindling seen. No consistent differences before and after 4½ G runs (1–3) but definite blocking of alpha after each 6-G exposure (runs 4–6).

Subject 10

Generally continuous alpha spindling of moderate intensity, with transient alpha blocking noter after runs 5 and 6 at 4½ G.

Subject 11

Strong alpha activity at all times, indicating a relaxed state with little alpha blocking due to arousal. No differences prerun or postrun, or between Gz force levels.

Patterns of blocking of large amplitude alpha waves reflect the subjective response of each individual to the experimental experience. Subjects 3 and 11 were able to maintain a consistently relaxed state before and after all runs.

Subjects 4, 5, 8, 9 and 10 were in a consistently relaxed condition at some times, and showed transient alpha blocking at others, without ever entering a fully aroused alert state. For Subjects 4 and 5, these epochs of partial arousal preceded runs 4–6 at 4½ G, for Subjects 9 and 10, they followed runs 4–6 at 6 and 4½ G respectively. For Subject 8, partial alpha blocking preceded all runs. These episodes of transient alerting seem based on each subject's personal appraisal of potentially threatening portions of the experimental protocol.

Beta Activity (13–20 Hz) (Table 4)

Intensity levels in this frequency band were generally unchanged after +4½ G exposures, and slightly but consistently elevated after 6-G exposure. This pattern was observed in Subjects 3, 5 and 6 and, with an increase of lesser amplitude after 4½-G exposure, in Subjects 8 and 9 as well. Subjects 10 and 11 showed no changes after any exposures, but these subjects were among the lowest resting intensity levels in the group and appeared devoid of any sinusoidal beta-range activity in the paper tracings. The other subjects showed some spindling in the beta range interspersed with either alpha material or very fast, low-voltage background activity. The increase in postrun beta activity was attributed almost wholly to an increase in fusiform EEG activity, despite the possible sensitivity of this band to EMG infiltration. The immed-

iately adjacent eyes-open epochs generally showed lower intensities. In addition, the 20–32 Hz band, theoretically more sensitive to EMG infiltration, often did not show parallel increases over the same epochs. The increases noted were due to both increased peak-to-peak amplitude of beta activity, and increased "on-time" of sinusoidal beta waves seen in the paper charts.

The post-6 G epochs all showed higher intensity values than the corresponding post-4½-G epochs, again with the exception of Subjects 10 and 11, who showed no consistent changes of any kind. These increases were entirely independent of the sequence of 4½ and 6 G exposures. Although exposure to +Gz forces did enhance beta intensities, the levels attained were entirely within normal limits, and no indications of pathological activity can be inferred.

Subject 4 was distinct from all others, in that he often displayed a high amplitude, sinusoidal dominant activity at 17 Hz which had all the characteristics of classical alpha. This dominant rhythm was subject to "blocking." A comparison with table 3 shows that beta and alpha intensities were equal for this subject and fluctuated in a precisely parallel manner.

Beta II (21–32 Hz) (Table 5)

Activity in this band was of low intensity with eyes closed, usually under 1 μv^2/sec/Hz, and reflected much less change after +Gz exposure than the 13–20 portion of the beta band. Only Subjects 6 and 9 showed any trend to higher values during +6 G runs, and the differences are neither large (0.1 to 0.25 μv^2, 30–40% of baseline) nor consistent. There was a distinct trend towards slightly higher levels post-run at both Gz levels, but the differences are extremely small (averaging 0.05–0.1 μv^2, 10% of baseline) and neither consistent nor significant for any one subject. For all 48 runs, all subjects combined, we noted 29 post run increases, 8 no changes and 11 net loses. The increases may well be of myogenic origin, but seem scattered equally through both G levels and sequences. The eyes-closed levels were at or below eyes-open values.

Subject 4 is again idiosynchratic, showing an intensity 10 times normal, which was attenuated (or "blocked") by opening the eyes. This unique subject appeared to generate "rhythms" in the 21–32 Hz band with characteristics similar to alpha activity, with prominent spindling initiated by eye-closure at rest.

EYES-OPEN/ACCELERATION DATA

During these epoches, the EEG paper chart tracings were obscured by high amplitude activity of myogenic origin, as in figure 1. Our interpretation of calculated narrow-band intensity values was necessarily made without reference to the ink-written charts for confirmation of the enhancement or absence of EEG wave-components or pathological signs.

Data for the 1–3 Hz band was discarded because of the difficulty of separating head movements

and spasmodic forehead contractions from low frequency EEG. Such contractions were almost continuous at peak G levels (associated with periodic straining to reduce loss of vision) and periods free of such artifacts could not be constructed, even with reference to video-taped records of facial movements. We noted that 1–3 Hz intensity at peak G was about 10 times greater than at rest, and showed individual-specific changes in activity between acceleration/deceleration epochs and peak epochs, rather than a series of intensities proportional to the profile of imposed Gz forces.

The resting, eyes-open epochs (II and VI) immediately preceding and following +Gz exposures were characterized by sporadic eye movements, which dominated the 1–3 Hz frequency band. No consistent patterns of post-exposure changes were observed among the several subjects.

Our analysis was concentrated on the two bands defined between 4 and 12 Hz, where myoelectric infiltration was predicted to be minimal. It was possible, particularly in the 4–7 Hz band, to demonstrate that certain epochs recorded at peak Gz levels were in fact free of activity of nonneural origin. Interpretation of data in other cases was based on a comparison with intensities within other bands, where the waxing and waning of myogenic activity could be determined with relative precision. Our parallel studies on the intensity spectrum of the scalp myogram (ref. 19) gave us guidelines for estimating the likely infiltration of EMG activity across adjacent frequency bands at various levels of contracting force and fatigue.

Both the 13–20 Hz and 21–32 Hz bands were dominated by myogenic activity during exposure to +Gz forces. We noted that spectral intensity in these two bands was proportional to the imposed G-force profile in most instances, the exceptions being epochs in which exceptionally heavy, paroxysmal whole body contractions occurred, which doubled or tripled the usual peak-G levels of intensity.

During peak-Gz force, intensity levels in the 21–32 Hz band reached values in excess of 100 times resting levels; the 13–20 Hz band reached values in excess of 10 times resting levels. Proportional intensity between these two bands exhibited the expected downward shift of overall scalp EMG spectra with fatigue. For this reason, shifting intensities in the 13–20 Hz band were closely considered when interpreting events in the 4–12 Hz domain. It was assumed that activity of myogenic origin in the 4–12 Hz region would remain in some stable proportion to shifts in 13–20 Hz levels, and that activity which waxed or waned in a manner contrary to the 13–20 Hz "reference" could be assigned a nonmyogenic origin.

Since proportional changes between the 4–7 Hz, 8–12 Hz and 13–20 Hz bands were the basis of our analysis of peak-acceleration EEG data, and since there were considerable individual differences in EMG intensity levels (attributable to individual differences in muscle contraction effort and fatigue), this data was tabled independently for each individual tested in tables 6–13.

Many subjects displayed an assymmetrical 13–20 Hz intensity profile, with higher levels of activity during deceleration than during acceleration to peak Gz force. This was due to an actual increase in muscular contraction levels, as reported by the subjects following a run and as monitored by TV. These effects were particularly considered and discounted before a neural origin was assigned to activity in other bands during the deceleration phase.

Theta (4–7 Hz) (Tables 6–13)

Three distinct patterns of theta activity enhancement during +Gz exposure were observed.

Subjects 4, 8, 11

Subjects 4, 8, and 11 produced peak values of theta intensity coincident with peak +Gz levels, with postrun values for runs 4–6 at or below pre-run values for runs 1–3. Theta intensities at +6 G exposures were usually higher than at +4½ G. During the +4½ G profile, Subjects 8 and 11 did not display theta intensities above their resting, eyes open levels, and the enhancement noted at +6 G immediately subsided when the +Gz force ceased. As the enhancement of theta intensity during Epochs III, IV and V of the +6 Gz profile for these two subjects was roughly proportional to the enhancement of intensity in the 13–20 Hz band, known to be more sensitive to activity of myogenic origin (table 4), the transient enhancement of intensity during +6 G exposure was tentatively assigned to myogenic origins within the theta band also.

For Subject 4, the increase in theta intensity was not proportional to increases in the 13–20 Hz band and, in fact, was higher at +4½ G than at +6 G. This activity was tentatively assigned a neuroelectric origin, but as the enhancement did not persist into the postrun period its nature cannot be more explicitly documented.

Subjects 3, 6, 10

Subjects 3, 6 and 10 produced peak values of theta intensity during the deceleration (V) or immediate postrun epochs (VI) of the acceleration profilts. Intensity values for epoch V (deceleration) exceeded values for epoch IV (peak +Gz force) for all three subjects during the +4½ Gz profiles, whereas during the +6 G runs, epoch IV (peak) values more nearly equaled the levels obtained during deceleration.

Assuming that signals of myogenic origin were wideband, or at least did not peak in the 4–7 Hz band, it is plausible to argue for a mixed representation of nervous and muscular activity in the +6 G epochs, with proportions that shift between peak and deceleration epochs. Activity during deceleration from +6 G in the 13–20 Hz band is no more than half the +6 G peak epoch values for all three subjects, while 4–7 Hz intensity remains equal, or slightly higher. Since all three subjects showed an enhancement of theta activity during deceleration from +4½ G peaks, it seems reasonable to assume that the same process occurred during deceleration from +6 G peaks as well, though masked by EMG infiltration during peak +Gz epochs. Subject 10 did not display this EMG infiltration, and the pattern is clear in both +4½ G and +6 G runs; indeed, levels of theta activity are higher during the +4½ G runs, although this was not true in frequency bands more sensitive to myogenic activity. The enhanced intensities observed during deceleration in these subjects occasionally persisted into the immediate postrun epoch, but could not be detected in the subsequent eyes-closed epochs extracted some 30 seconds later.

Subjects 5 and 9

Subjects 5 and 9 exhibited a similar pattern but with enhanced activity in the 4–7 Hz band during the acceleration (epoch III) period as well as deceleration (epoch V). This pattern was clearly displayed only during the +4½ G profiles. During +6 G runs higher levels of activity during peak or deceleration epochs obscured this effect. Here, too, one can interpret the data as a confounding of two effects, an increase during acceleration and deceleration of neurodenic origin, and an increase during peak Gz (and continuing during deceleration) of myogenic origin.

No effects were observed attributable to the sequence in which the +4½ and +6 G runs were accomplished.

Alpha (8–12 Hz) Band (Tables 6–13)

Interpretation of spectral intensity in the 8–12 Hz band during +Gz exposure was contingent on the extent of alpha-blocking observed in each subject upon opening the eyes before each run, and upon the actual initiation of the +Gz profile.

Subjects 4, 5, 6, 9, 10 and 11 produced a complete blocking of resting alpha upon opening the eyes the residual intensities in the 8–12 Hz band being compatible with occasional desynchronized isolated wave forms. No further enhancement of arousal, with concomitant attenuation of 8–12 Hz intensity, would be expected during subsequent epochs, whatever the potential "alerting" value of the +Gz exposures. Since this blocked, aroused state was also demonstrably present in the ink-written record immediately after the cessation of masking EMG components at the end of a run, we have assumed that any enhancement of alpha intensities in these subjects during the +Gz exposures was of a neural origin distinct from the mechanism of alpha-enhancement which accompanies a relaxation of arousal. Such enhancement of 8–12 Hz activity did occur in these subjects, in a manner that eliminated a possible myogenic origin for at least some of the observed increase in intensity. Any increases in intensity proportional to either +Gz force or the predominantly myogenic activity in the 21–32 Hz band were assumed to be of nonneural origins. The observed exceptions are noted below.

+ 4½ G Levels

Only Subject 10 showed an increase in 8–12 Hz intensity throughout +Gz exposure which excedes the power levels in the 13–21 Hz band. Some other subjects display the anticipated intensity ratio of at least 2:1 between beta and alpha intensities suggested by spectral analysis of predominantly myogenic activity.

+ 6 G Levels

Subjects 4, 5, 9 and 10 showed an increase in 8–12 Hz intensity during the deceleration (epoch V) phase of the +Gz exposure profile which approached or exceded parallel spectral intensities in the 13–21 Hz band. By contrast, 8–12 Hz intensities during the acceleration and peak phases (epochs III and IV) were proportionally much lower than 13–21 Hz levels. Subjects 6 and 11 did not display such disproportional 8–12 Hz intensity during the deceleration phase.

Subjects 3 and 8 were more difficult to interpret, since they presented an incompletely alpha-blocked record with eyes open, which was further blocked when the +Gz increments actually began in epoch III. These records did show alpha spindling with eyes open, which presumably disappeared during epoch III, since a lower level of spectral intensity was observed, despite the infiltration of EMG activity which effectivey masked the paper tracing. It was not possible to determine whether the enhanced intensity observed in Subject 8 over epoch V (both +4½ and +6 G runs), was analogous to the increases seen in Subjects 4, 5, 9 and 10, or simply due to a decrease in arousal during this late phase of the +Gz profile.

SINGLE, EXTENDED DURATION RUNS

At the time of this study, one member of the SAM centrifuge subject panel (Subject 4) was trained and qualified to withstand a continuous +7 G exposure for over 45 seconds without loss of central vision. Continuous EEG recordings were made during such a run, and continued for 10 minutes postrun. Spectral values for the EEG during this run are given in Table 14.

This data indicates no buildup of power in any frequency band during the extended exposure. In the 1–7 Hz domain power decreased between the first and third 15-second epochs; in the 8–30 Hz domain it was substantially unchanged. The reduction in 4–7 Hz power was certainly due to an attenuation of brain electrical output in that band, as the myogenic activity in the 13–32 Hz band was unchanged in sum and distribution. While this reduction in theta intensity cannot be assigned to a definite origin, clearly no aberrant or pathological processes were influencing EEG spectra during this 45-second exposure.

The eyes-open, resting spectra immediately before and after +Gz exposure were virtually identical. The slight enhancement of 13–20 Hz activity postrun was attributed to a diminution of the subject's alert state postrun; an idiosynchratic characteristic of this particular subject.

The first postrun eyes-closed epoch showed enhanced power in all bands when compared to prerun eyes-closed values. These intensity levels were all much reduced 4 minutes later, and were at or below prerun levels within 7 minutes. This broad-band enhancement of activity postrun was seen in most of the subjects studied in the short exposure protocol, though not to this degree. Here, too, no definite origin of this phenomenon can be determined, but the presence of a gradual, postrun, pathological low frequency buildup can be excluded.

An additional subject qualified for a 45-second +6½ G run, but EEG data during the actual exposure was lost when the electrode-skin contact broke during a violent straining maneuver. Prerun and postrun resting records and their calculated spectra from 0–32 Hz are given in figure 3. No significant changes appeared immediately postrun, although an increase in intensity at 1 Hz is present after 4 minutes. The total lack of increased intensity between 2 and 7 Hz precludes a pathological interpretation of this data.

SECTION V

DISCUSSION

The results reported above support two general conclusions: that the electrical activity of the brain is altered by exposure to +Gz forces, even in the absence of a visual blackout, and that this alteration is not pathological and not cumulative within the +Gz dimensions of our experimental protocol. Since our protocol induced many atypical cardiac events, the central nervous system appears to be relatively better protected against +Gz stress than is the circulatory system. Tissue compression and the separation of cellular particulate inclusions on a gravitational gradient, while potentially deleterious, do not seem to constitute limiting factors for human +Gz exposure under present conditions.

The exact mechanisms underlying the observed EEG changes cannot be specified with any certainty, and we must speculate as to their nature. Partial anoxia of CNS tissues would cause an enhancement of intensity in lower frequencies which would endure beyond the cessation of +Gz forces, and no such pattern of EEG changes was noted. Despite the unequivocal evidence of retinal circulatory impairment and transient anoxia, no parallel events seemed to have occurred in the cerebral tissues.

We suggest that the broadband enhancement of intensity seen in pre- and postrun resting records was a consequence of compensatory changes in peripheral blood vessels (such as increased tonus in vascular walls) during +Gz exposure, which increased the volume of blood flow beyond normal levels for a brief period following cessation of the +Gz force. Those subjects who showed no disparity of intensity changes between +4½ G and +6 G runs may be assumed to have reached a stable plateau of compensatory vascular changes under both conditions. Those subjects showing greater EEG intensity increases under +6 Gz conditions may be presumed to have a greater range compensatory circulatory modifications available (ref. 12).

The EEG changes noted during actual +Gz exposures also resist specification of a mechanism of origin. Tissue compression and its consequent oedema would persist into the postrun epochs and cause persistent changes in the theta band which were not observed. The individual variation in theta-band intensity enhancement which occurred at peak +Gz epochs for some subjects and during increments and decrements of +Gz force for others, suggests the influence of a subjective perception of environmental stressors. Both theta and alpha intensity are capable of increasing at moments of subjective or perceived stress (ref. 7) The progressive diminution of theta intensity during our one extended run is also compatible with a subjective origin of this activity.

Intense vestibular stimulation may also enhance theta activity (ref. 4) and may have had this effect on those subjects displaying theta peaks during increments and/or decrements of +Gz force. At these times the centrifuge gondola rotates in its gymbals as it swings freely to maintain a perpendicular +Gz vector, and the subjects experience the sensation of a climbing or diving turn. Motions of the head at these times will induce a vestibular perception of coriolis forces. We cannot exclude other origins for these effects, but we note again that the increased theta

intensities our subjects displayed were all well within the range of normal responses to stressor or vestibular stimuli and, whatever their origin, cannot be interpreted as indicating either a disruption of CNS physiological processes, or an impairment of perception or performance ability at the cerebral level.

The changes in EEG spectra we observed are compatible with many, but not all, of the gross observations on humans reported by other investigators. Gazenko (ref. 11) reported enhancement of theta and alpha waves during exposures in the +5 Gz range, and a return to normal eyes-closed alpha rhythms within 5 minutes. Sem-Jacobsen reported theta and delta bursts during +5–+7 Gz exposures in actual flight (ref. 21). Both reported prominent slow waves persisting after exposure, and Sem-Jacobsen recorded several episodes of unconsciousness. We attribute the persistent slow waves to the presence of corticle anoxia, associated with complete blackout. The straining maneuver and other +Gz tolerance training our subjects received seems to have allowed them to avoid total backout at +Gz levels where others could not.

Levels of theta and alpha activity are not specified numerically in these reports, but we presume the intensity enhancements we recorded in our present study are of considerably lower magnitude. Theta enhancement in a comparable amplitude range was noted in one of the Gemini astronauts during both liftoff (+Gx) and prolonged weightlessness (ref. 4), without slow wave bursts, and was attributed to an augmentation of orienting responses in an unusual vestibular environment. This is compatible with our present centrifuge recordings, and the more extreme theta increases observed by others are likely a consequence of anoxia and not an extension of stress or vestibular responses. A definite anoxic origin of high-intensity theta bursting in the cat and monkey has been demonstrated in centrifuge studies carried beyond the point of unconsciousness (refs. 2, 3).

The origin of theta enhancement contingent on the initiation of acceleration, and before peak levels are reached, is suggested by Gazenko's studies on rabbits with implanted electrodes in the reticular formation (ref. 11). Groups of neurons were recorded which responded immediately to the onset of acceleration with rhythms in the 5–6 Hz range. These persisted for 80–85 seconds during a continuous +5 Gz acceleration plateau, and then were extinguished. As acceleration force was diminished, the rhythms again appeared. Similar patterns of theta enhancement in deep structures contingent on alterations of angular acceleration have been reported in the cat (ref. 8). These patterns were paralleled in five of our eight subjects, those who showed higher theta intensities during increments or decrements of +Gz force than they showed at Peak +Gz loads.

Squires et al. (ref. 22) have reported a study very similar to our present investigation, in which however, the subjects were centrifuged past the point of blackout. Although many EEG changes were associated with the points of blackout and loss of consciousness, particularly 5–7 Hz intensity enhancement, some changes in spectra prior to blackout seem to have persistently occurred. Although their data is presented in percentage-increase format rather than absolute physical units, there does seem to be some evidence in their data for a "two-tiered" effect of 4–12 Hz intensity enhancement, with a slight increase attributable to perceptual and stressor effects preceeding a major increase attributable to anoxia and loss of consciousness. One of Squires' published figures also shows a double peak for intensities in the 8.5–11.5 Hz range, the two inten-

sity peaks coinciding in a rough way with the increment and decrement of +Gz force, with intensity falling off during the sustained +Gz force plateau.

Gillies (ref. 12) ascribes all important EEG changes during acceleration to anoxia, with the exception of alpha bursts coinciding with loss of central vision, and equivalent to closing of the eyes. This position seems to us to be untenable. The events associated with blackout and impaired consciousness are of greater magnitude, but consistent and quantifiable changes in the EEG occur in the absence of blackout in a changing G-force environment. As improved training and mechanical protection increase the G-force which can be sustained without experiencing blackout, the smaller scale changes in the EEG assume greater importance as indicators of CNS state in a G-force environment in which complex perceptual and performance tasks are to be performed. On present evidence, performance levels need not be compromised by the CNS changes occurring during +6 Gz and +7 Gz exposures where central vision is maintained.

The noncumulative nature of the smaller, sub-blackout type of G-force-contingent EEG changes is confirmed by Barer et al. (ref. 5) who reported percentage increases in EEG spectra during continuous 85-second exposures to +12 Gx. A 50% enhancement in the 8–13 Hz band was recorded unchanged from the onset of +Gx acceleration through the 85-second exposure period. No separate measurement of the 4–7 Hz band was made. There was no indication in this data of the gross (5×–10×) increases and decreases of alpha activity that are often contingent on blackout and loss of consciousness.

Three basic processes appear to influence the characteristics of the EEG during G-force exposures. One is contingent on absolute blood flow, effects blackout, loss of consciousness, and the very large magnitude EEG changes associated with these. Another is contingent on the vestibular stimuli associated with changes in the G-force environment and is reflected in transient changes in intensity which may appear in any of the frequency bands between 4 and 15 Hz. The third is contingent on the level of metabolic processes in CNS tissue and the mechanisms of compensation employed to counter hemodynamic impairment. This process is responsible for the long duration shifts in baseline intensities often observed on a broadband basis.

Subjective perception of body orientation and stress also effects transient EEG levels, and these effects may be superimposed on the three processes listed above. Any hope of distinguishing these various effects rests on a precise quantification of the EEG in absolute physical units, since neither cursory examination of paper-tracings nor frequency analysis expressed in baseline percentage shifts is sufficiently precise for this purpose. A similar quantification is also necessary for those frequency bands in which EMG activity predominates, if the extent of infiltration of EMG activity into the slower frequency EEG domain is to be estimated.

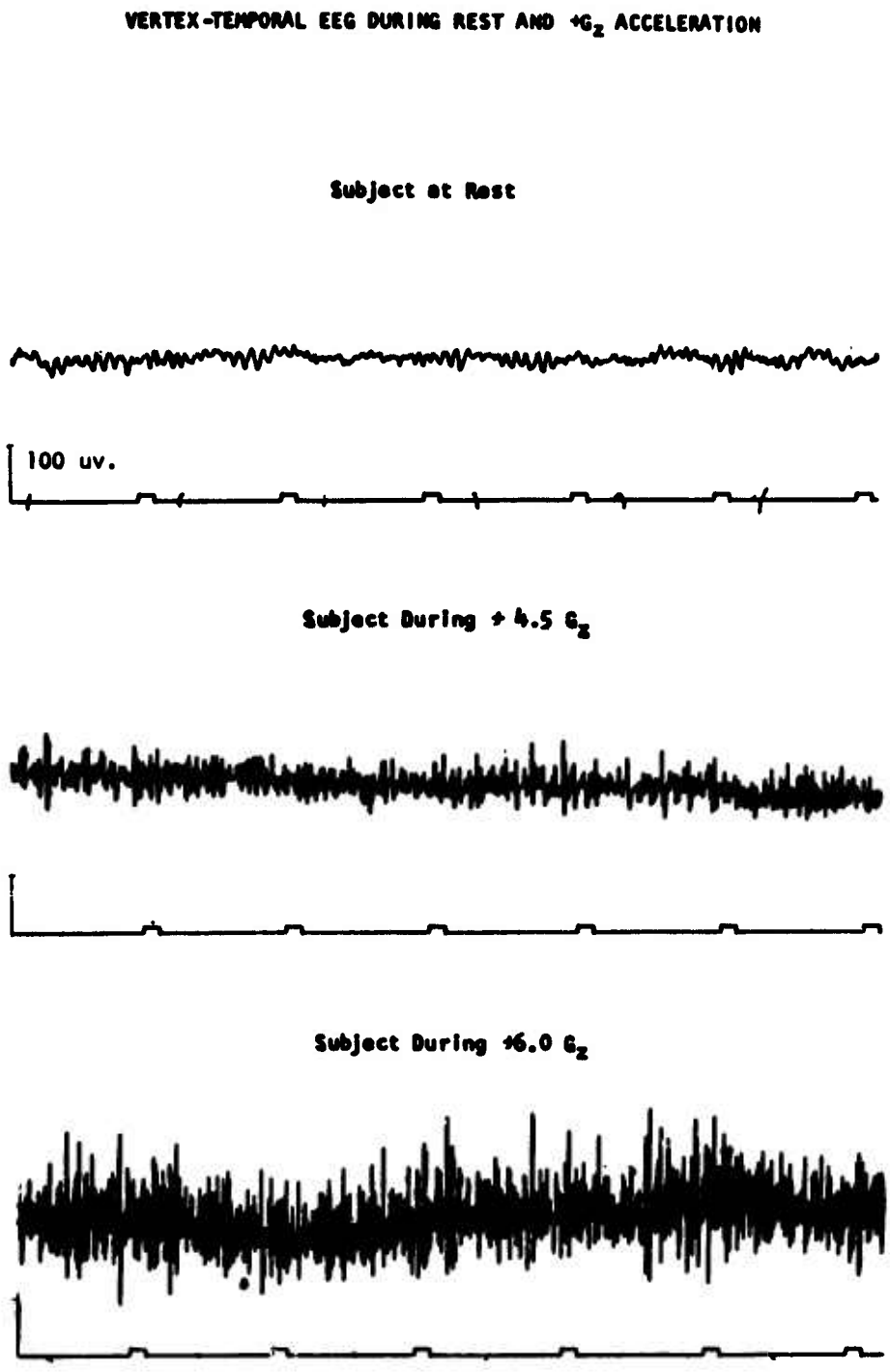

Figure 1. Sample EEG Chart Records Showing The Differential Infiltration Of Myogenic Activity Between +4½ Gz And +6 Gz Exposure Levels. One-second time scale appears under each sample.

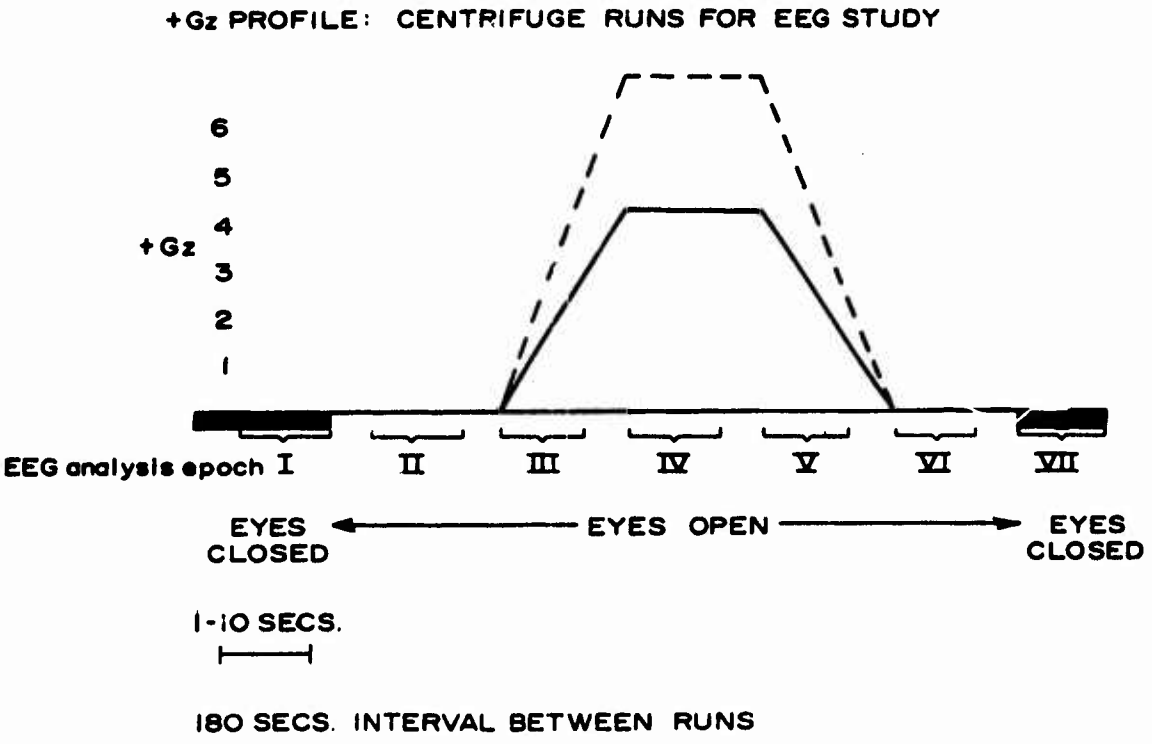

Figure 2. Exposure Profile For Experimental Acceleration Force. Epochs defined for EEG analysis are indicated.

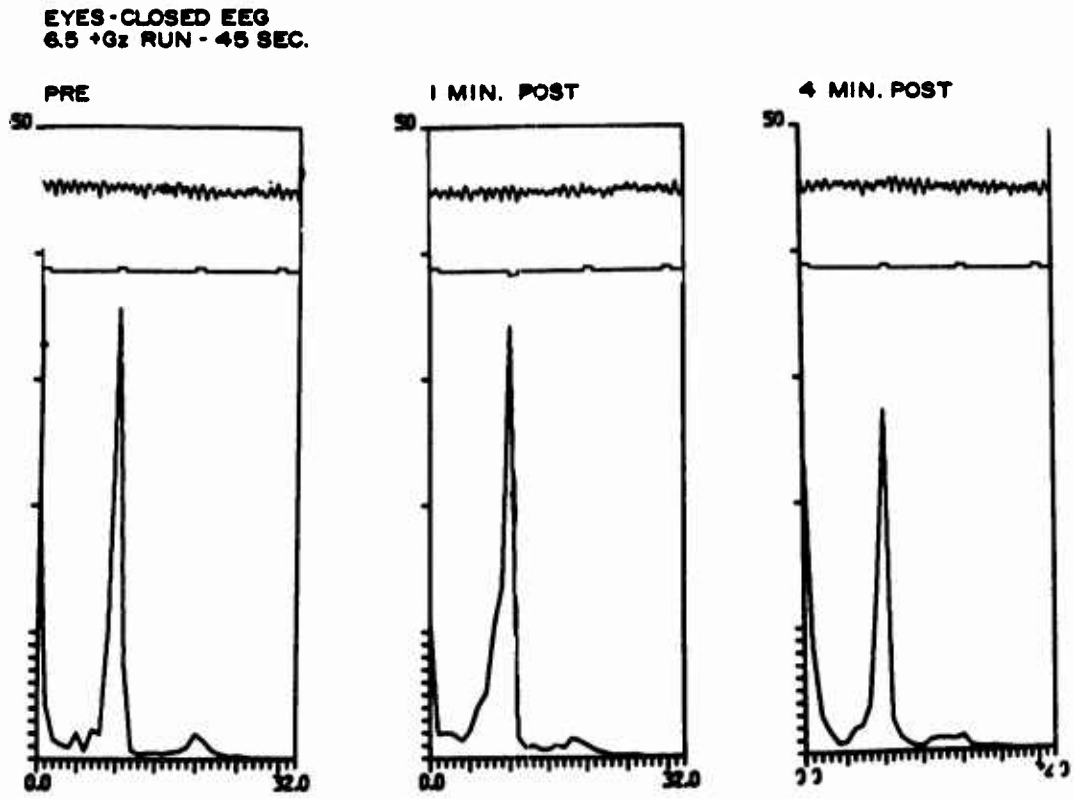

Figure 3. Prerun And Postrun Spectral Profiles For An Extended, 45-Second +6½ Gz Exposure. Profiles are normalized. Slight increase in power at 1 Hz noted 4 min post.

TABLE 1. EYES-CLOSED RESTING DATA (1 TO 3 Hz). LPO CHANNEL EXCEPT SUBJECT 10 (RPO). Averages of three runs. "Differences" indicate direction of intensity change in each individual run contributing to the average. Changes of less than 5% are scored equal (=). Values in μv^2/sec/Hz.

FREQUENCY BAND 1 TO 3 HZ

	SUBJECT	3	5	9	11	4	6	8	10
			+4½ Gz Runs				+6 Gz Runs		
Runs 1–3	Epoch I	53.0	14.4	14.0	5.7	23.0	4.7	9.5	7.6
	(Differences)	− − −	− + +	− + −	− − +	− + +	+ + −	+ + +	+ + +
	Epoch VII	47.0	13.0	12.9	5.6	26.7	6.1	17.4	10.0
			+6 Gz Runs				+4½ Gz Runs		
Runs 4–6	Epoch I	34.0	11.4	17.1	11.0	20.2	5.5	10.2	6.7
	(Differences)	+ + +	− + +	+ − −	− − +	+ − −	− + =	+ + +	+ − −
	Epoch VII	54.0	12.8	17.7	11.4	20.3	4.7	15.4	5.7

TABLE 2. EYES-CLOSED RESTING DATA (4 TO 7 Hz). LPO channel except Subject 10 (RPO). Averages of three runs. "Differences" indicate direction of intensity change in each individual run contributing to the average. Changes of less than 5% are scored equal (=). Values in μv^2/sec/Hz.

FREQUENCY BAND 4 TO 7 HZ

	SUBJECT	3	5	9	11	4	6	8	10
			+4½ Gz Runs				+6 Gz Runs		
Runs 1–3	Epoch I	15.0	3.3	3.0	2.5	10.6	1.3	2.8	3.0
	(Differences)	+ + +	− − +	+ + +	− − +	+ + −	− + +	+ + +	− + +
	Epoch VII	21.0	2.7	3.4	2.1	10.9	1.4	4.8	3.1
			+6 Gz Runs				+4½ Gz Runs		
Runs 4–6	Epoch I	13.0	2.2	4.1	2.1	5.3	1.2	2.8	2.6
	(Differences)	+ − +	+ + −	+ − +	+ − =	+ + +	= + +	+ + +	= − =
	Epoch VII	20.0	2.8	4.4	1.9	9.2	1.6	6.9	2.5

TABLE 3. EYES-CLOSED RESTING DATA (8 TO 12 Hz). LPO channel except Subject 10 (RPO). Averages of three runs. "Differences" indicate direction of intensity change in each individual run contributing to the average. Changes of less than 5% are scored equal (=). Values in μv^2/sec/Hz.

FREQUENCY BAND 8 TO 12 HZ

	SUBJECT	3	5	9	11	4	6	8	10
			+4½ Gz Runs				+6 Gz Runs		
Runs 1–3	Epoch I	88.0	6.1	10.5	24.3	16.0	2.8	18.4	21.1
	(Differences)	− − +	− − −	+ + −	− + +	+ + −	− + +	+ + +	+ − =
	Epoch VII	90.5	4.3	12.1	26.8	18.8	3.3	70.8	20.0
			+6 Gz Runs				+4½ Gz Runs		
Runs 4–6	Epoch I	82.5	3.3	16.7	28.0	8.0	1.8	33.0	21.3
	(Differences)	+ − +	+ + +	− − −	+ − +	+ + +	+ + −	+ + +	+ − −
	Epoch VII	95.0	5.3	9.1	24.6	18.0	2.0	60.0	15.2

TABLE 4. EYES-CLOSED RESTING DATA (13 TO 20 Hz). LPO channel except Subject 10 (RPO). Averages of three runs. "Differences" indicate direction of intensity change in each individual run contributing to the average. Changes of less than 5% are scored equal (=). Values in μv^2/sec/Hz.

Frequency Band:

	SUBJECT	3	5	9	11	4	6	8	10
			+4½ Gz Runs				+6 Gz Runs		
Runs 1–3	Epoch I	3.6	4.9	1.9	1.5	19.0	0.7	1.6	1.3
	(Differences)	− + −	− + =	− + +	− − =	+ = −	+ + +	+ + +	− + =
	Epoch VII	3.6	4.9	2.2	1.3	20.0	1.2	3.5	1.5
			+6 Gz Runs				+4½ Gz Runs		
Runs 4–6	Epoch I	2.9	3.0	2.6	1.3	8.0	0.7	2.0	1.9
	(Differences)	+ + +	+ + +	− + +	+ − +	+ + +	= + −	+ + =	− − −
	Epoch VII	4.3	5.2	2.9	1.3	12.0	0.7	3.0	1.2

TABLE 5. EYES-CLOSED RESTING DATA (21 TO 32 Hz). LPO channel except Subject 10 (RPO). Averages of three runs. "Differences" indicate direction of intensity change in each individual run contributing to the average. Changes of less than 5% are scored equal (=). Values in μv^2/sec/Hz.

FREQUENCY BAND 21 TO 32 HZ

	SUBJECT	3	5	9	11	4	6	8	10
			+4½ Gz Runs				+6 Gz Runs		
Runs 1–3	Epoch I	.39	.40	.72	.40	4.68	.26	.31	.40
	(Differences)	− + +	+ = +	− + +	+ − +	+ + +	+ + +	+ − +	− + =
	Epoch VII	.46	.50	.74	.46	5.60	.35	.31	.40
			+6 Gz Runs				+4½ Gz Runs		
Runs 4–6	Epoch I	.37	.34	1.06	.37	5.20	.15	.24	.40
	(Differences)	= + +	+ − =	− = +	+ = −	+ + −	+ + +	+ + =	− − =
	Epoch VII	.44	.51	1.20	.44	6.40	.24	.33	.30

TABLE 6. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 3). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 3

Frequency Band:		4–7	8–12	13–20
	Epochs:			
Runs 1–3	II (Rest)	3.4	6.69	1.54
(+4½ Gz)	III (Increasing G-Force)	2.8	3.58	2.42
	IV (Peak G-Force)	3.6	5.56	9.44
	V (Decreasing G-Force)	4.9	6.94	4.17
	VI (Rest)	6.7	6.99	2.63
Runs 4–6	II (Rest)	3.0	7.77	2.05
(+6 Gz)	III (Increasing G-Force)	4.5	8.05	7.69
	IV (Peak G-Force)	7.5	31.69	59.49
	V (Decreasing G-Force)	7.3	13.01	9.52
	VI (Rest)	3.3	4.85	1.78

TABLE 7. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 4). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 4

Frequency Band		4–7	8–12	13–20
	Epochs:			
Runs 1–3	II (Rest)	6.1	3.34	2.00
(+6 Gz)	III (Increasing G-Force)	10.3	5.29	5.44
	IV (Peak G-Force)	11.9	8.53	22.55
	V (Decreasing G-Force)	10.0	8.65	10.80
	VI (Rest)	7.8	3.90	3.27
Runs 4–6	II (Rest)	7.8	2.97	2.15
(+4½ Gz)	III (Increasing G-Force)	8.2	4.30	2.52
	IV (Peak G-Force)	14.1	5.55	6.39
	V (Decreasing G-Force)	7.5	4.93	4.80
	VI (Rest)	8.7	3.71	2.62

TABLE 8. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 5). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 5

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	2.6	1.82	2.32
(+4½ Gz)	III (Increasing G-Force)	3.2	2.59	3.64
	IV (Peak G-Force)	2.7	6.11	11.39
	V (Decreasing G-Force)	3.2	2.83	3.07
	VI (Rest)	2.8	2.12	2.53
Runs 4–6	II (Rest)	2.1	1.94	1.72
(+6 Gz)	III (Increasing G-Force)	2.9	2.52	3.99
	IV (Peak G-Force)	4.1	8.26	21.98
	V (Decreasing G-Force)	5.0	5.59	5.34
	VI (Rest)	3.8	2.03	2.64

TABLE 9. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 6). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 6

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	3.4	2.57	1.47
(+6 Gz)	III (Increasing G-Force)	3.2	3.94	5.50
	IV (Peak G-Force)	4.1	7.25	16.32
	V (Decreasing G-Force)	4.2	5.07	8.73
	VI (Rest)	4.2	4.86	3.98
Runs 4–6	II (Rest)	3.1	1.91	0.64
(+4½ Gz)	III (Increasing G-Force)	2.5	3.42	3.76
	IV (Peak G-Force)	2.8	6.19	6 63
	V (Decreasing G-Force)	3.2	3.51	4.25
	VI (Rest)	4.2	2.77	1.49

TABLE 10. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 8). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 8

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	3.5	24.30	1.07
(+6 Gz)	III (Increasing G-Force)	3.8	2.90	2.90
	IV (Peak G-Force)	8.2	3.84	7.66
	V (Decreasing G-Force)	4.4	10.82	2.21
	VI (Rest)	4.7	4.40	1.60
Runs 4–6	II (Rest)	5.0	4.36	1.86
(+4½ Gz)	III (Increasing G-Force)	3.3	2.54	1.68
	IV (Peak G-Force)	3.6	7.60	3.16
	V (Decreasing G-Force)	3.2	10.49	1.68
	VI (Rest)	3.4	10.90	1.02

TABLE 11. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 9). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 9

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	7.2	3.19	1.58
(+4½ Gz)	III (Increasing G-Force)	9.8	5.53	4.87
	IV (Peak G-Force)	6.4	5.32	6.17
	V (Decreasing G-Force)	8.1	6.42	4.64
	VI (Rest)	6.5	4.02	2.19
Runs 4–6	II (Rest)	8.0	3.07	1.86
(+6 Gz)	III (Increasing G-Force)	9.2	3.25	5.54
	IV (Peak G-Force)	11.0	7.25	15.30
	V (Decreasing G-Force)	6.3	6.91	6.81
	VI (Rest)	9.9	3.50	2.21

TABLE 12. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 10). Averages of three runs. Values in μv^2/sec/Hz. RPO channel.

SUBJECT NO. 10

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	3.3	4.00	2.27
(+6 Gz)	III (Increasing G-Force)	4.8	10.56	6.48
	IV (Peak G-Force)	4.7	19.46	25.83
	V (Decreasing G-Force)	7.3	16.67	13.87
	VI (Rest)	3.9	3.83	2.44
Runs 4–6	II (Rest)	2.1	3.77	2.53
(+4½ Gz)	III (Increasing G-Force)	2.9	5.47	8.99
	IV (Peak G-Force)	4.1	14.70	11.89
	V (Decreasing G-Force)	5.0	17.88	5.44
	VI (Rest)	3.8	4.80	1.98

TABLE 13. EYES-OPEN ACCELERATION DATA (SUBJECT NO. 11). Averages of three runs. Values in μv^2/sec/Hz. LPO channel.

SUBJECT NO. 11

Frequency Band		4–7	8–12	13–20
	Epochs:			
(Runs 1–3)	II (Rest)	1.6	2.60	0.81
(+4½ Gz)	III (Increasing G-Force)	1.9	3.30	2.67
	IV (Peak G-Force)	2.3	4.08	8.32
	V (Decreasing G-Force)	1.9	2.98	4.54
	VI (Rest)	1.4	6.70	1.27
Runs 4–6	II (Rest)	2.4	2.57	1.02
(+6 Gz)	III (Increasing G-Force)	5.2	5.20	12.25
	IV (Peak G-Force)	10.2	19.00	48.63
	V (Decreasing G-Force)	4.7	7.26	9.51
	VI (Rest)	2.3	5.66	1.36

TABLE 14. EXTENDED DURATION + 7 Gz RUN (SUBJECT NO. 4). Values in μv^2/sec/Hz. Values for one channel (LPO) given.

Frequency Band	1–3	4–7	8–12	13–20	21–32
Eyes Closed, Prior to Run.	20.2	5.3	16.0	12.0	4.6
Eyes Open, Prior to Run.	26.6	7.3	5.0	1.7	1.1
First 15 secs, +7 Gz Peak.	304.0	24.3	30.8	74.1	45.5
Second 15 secs, +7 Gz Peak.	252.0	14.7	27.3	65.0	40.3
Third 15 secs, +7 Gz Peak.	123.0	15.4	26.9	66.8	41.2
Eyes Open, Post Run.	30.3	6.6	4.6	4.7	2.0
Eyes Closed 1 min Later.	31.0	16.3	29.9	31.6	3.7
Eyes Closed, 4 min Later.	21.7	7.7	21.2	15.2	6.5
Eyes Closed, 7 min Later.	18.2	6.1	14.8	15.1	5.5

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